

Traumatic pancreatic rupture occurring during a convulsive crisis. Case-report and review in literature

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Abstract. Introduction. Abdominal injuries are very common in modern society. Their aetiology is increasingly diverse, with road accidents occupying the first place by far. Abdominal traumas that occurring during convulsive crisis are exceptional, and grade III pancreatic injuries during convulsive crisis are rare, which is why we decided to present this case. **Case report.** We present the case of a male patient 70 years-old, weighing 72kg, with a height of 174cm, and robust physique. The patient was brought by ambulance to the Emergency Department with generalized abdominal pain accompanied by generalized muscle contracture, severe hypotension (65/35mmHg), and tachycardia (140bpm). These symptoms appeared after an abdominal trauma that occurred approximately 5-6 hours earlier during a convulsive crisis, which resulted in a fall and impact on the anterior abdominal wall. The local clinical examination revealed a traumatic mark on the right frontal area (approximately 65/10mm ecchymosis), an abdominal wall hematoma causing deformation, and ecchymosis on the upper abdominal floor extending to the right hypochondrium. A FAST-ultrasound was performed, which revealed a large amount of free fluid with a transonic appearance, slightly heterogeneous. **Discussions.** From an epidemiological perspective, we are dealing with a rare, trauma, with an incidence that varies according to statistics but generally does not exceed 10% of abdominal traumas. In terms of aetiology, as mentioned above, these injuries can be caused by abdominal contusion or penetrating injuries. When considering surgical intervention, the correct approach must take into account Ballinger's Rule of 3 R. Possible postoperative complications that can occur in patients with such injuries include acute necrotic-haemorrhagic pancreatitis, local abscess formation, pancreatic fistula, posttraumatic pseudocyst, and pulmonary thromboembolism. **Conclusions.** In emergency surgical practice, we encounter extremely diverse situations, but traumatic pancreas injuries are particularly rare and challenging to diagnose. Many surgeons may not encounter a pancreatic traumatic injury throughout their entire career (and in fact, they wouldn't want to). Although traumatic injuries to the pancreas occur in a small percentage of cases, they should not be overlooked and should be considered within the spectrum of possible injuries.

Key words: pancreatic trauma, hemoperitoneum, Ballinger rules, necrotic pancreatitis.

Introduction.

Abdominal traumas are very common in modern society. Their aetiology is increasingly diverse, with road accidents, in their various forms, occupying the first place by far. Abdominal traumas occurring during convulsive crises are exceptional, especially with such manifestations and violence. I couldn't find any bibliographic references that address this subject.

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Grade III pancreatic traumas within convulsive crises are rare, which is why I decided to present this case even though preoperative imaging documentation was not possible. The first case of pancreatic trauma was presented in 1827 by Travers in London and was published in *The Lancet*. In 1882, Kulenkampff presented the first case of a patient who survived pancreatic trauma and later developed a pancreatic pseudocyst. In 1903, Kocher presented the surgical manoeuvre that bears his name, which is still used today and provides excellent visibility of the pancreatic head (1).

The frequency of pancreatic traumas ranges from 0.2% to 6% in some statistics, and even up to 10% of all abdominal traumas, but those with partial or complete rupture are much rarer. The pancreas, being a retroperitoneal organ and somewhat protected by the other intraperitoneal organs, has a lower incidence of trauma (1, 2, 3, 4). The frequency of complications in this pathology is very high, ranging from 60% to 93% (4), and the mortality rate is between 20% and 40% (4), and in various statistics, it can even reach 50% (5).

Case report

We present the case of a 69-year-old male patient from a rural area who was brought to the emergency department of the hospital by ambulance around 6:45 PM. He complained of generalized abdominal pain with a tendency for generalized muscle contraction, severe hypotension (65/35 mmHg), and tachycardia (140/min). These symptoms occurred after an abdominal trauma that occurred approximately 5-6 hours ago during a seizure with a forward fall and impact on the anterior abdominal wall. The patient was confused and had no recollection of what had happened. It should be noted that the family found him around 6:15 PM after a period of time (exact time unknown: they had a meal together around 12:30 PM) following the seizure.

During the clinical examination, the patient, weighing 72 kg and measuring 174 cm in height, was observed to have pale, sweaty, and cold skin. His blood pressure was 65/35 mmHg, and his pulse rate was 140/min. Local examination revealed generalized abdominal

muscle contracture, which prevented palpation of the abdomen. Additionally, a right frontal traumatic mark (approximately 65/10 mm bruise), abdominal wall hematoma causing deformation, and upper abdominal bruising extending towards the right hypochondrium were observed.

A focused assessment with sonography for trauma (FAST) ultrasound was performed in the emergency department on the uncooperative patient, which revealed the presence of a dilacerating abdominal wall hematoma extending towards the right hypochondrium, obstructing visible access to the peritoneal cavity. A large amount of fluid was observed in the peritoneal cavity, and the spleen was visualized with an intact capsule surrounded by fluid. The liver appeared heterogeneous in the left lobe, making it difficult to visualize, and the pancreas was also difficult to visualize but appeared intact. Diffuse contouring of the spleen and free fluid with transonic content and slight inhomogeneity were observed in the peritoneal cavity. Peritoneal aspiration was performed, and blood was extracted, confirming the presence of hemoperitoneum.

Therefore, the following diagnoses were established upon admission: **Hemoperitoneum. Haemorrhagic shock. Severe abdominal contusion. Cranio-cerebral trauma. Status post-seizure.**

The patient underwent surgical intervention under general anaesthesia with endotracheal intubation and mechanical ventilation. The patient had a high anaesthetic risk with an ASA classification of 4. A xiphoid-umbilical laparotomy was performed, which was extended subumbilically and then branched to the right due to poor visibility. Exploration of the abdominal cavity revealed a dilacerating wall hematoma, massive hemoperitoneum (2200 mL was aspirated, and approximately 500 mL was absorbed by the surgical field), complete dilacerating rupture of the pancreas located on the vertebral column relief to the left of the superior mesenteric artery, significant crushing of the pancreatic tissue over a length of approximately 3-4 cm, with severed and bleeding peripancreatic vessels that were

ligated. A retroperitoneal hematoma fused towards the right up to the hepatic flexure of the colon and towards the left up to the splenic flexure, extending from the celiac trunk to the pelvis, with infiltration of the mesentery and postoperative peritoneal adhesions, some of which led to significant depolarization and required enterorrhaphy. Therefore, the evacuation of the peripancreatic hematoma and partial evacuation of the retroperitoneal hematoma, drainage of the hemoperitoneum, lysis of adhesions, enterorrhaphy in 2 planes, haemostasis by ligating the bleeding pancreatic and peripancreatic vessels, debridement of devitalized peripancreatic areas, detachment of the duodenojejunal angle, pancreatic suturing, thorough lavage, control of haemostasis, and complete reconstruction of the abdominal wall were performed.

The patient was transported intubated to the intensive care unit (ICU), where care and monitoring continued. Along with hydro-electrolyte and acid-base balance correction, nutritional and energy support, the treatment regimen included dual antibiotic therapy (initially Cefort, later changed to Meronem and Metronidazole), anticoagulants, potent analgesics, pancreatic anti-oedema agents, transfusions for vascular bed restoration, and ongoing monitoring of vital parameters. Additionally, the patency of the drainage tubes, temperature curve, and daily laboratory analyses were closely monitored.

Once the patient achieved cardiovascular stability and a cranio-thoraco-abdomino-pelvic CT scan became possible, further exploration was conducted. The CT scan revealed the following findings: a discreet subcortical left frontal hyperdense area measuring approximately 17/18 mm with mild perilesional vasogenic oedema at the cerebral level; a right basal posterior pulmonary iodophilic consolidation with air bronchogram at the thoracic level; and posttraumatic bilateral pleurisy at the pleural level. In the left hepatic lobe segment II, two hypodense areas measuring 12 mm and subcapsular measuring 6 mm were identified, possibly indicating a path of dilaceration. At the level of the abdominal

aorta, a dissection of the abdominal aorta from the origin of the superior mesenteric artery with an extension of approximately 30 mm to the emergence of the left renal artery, classified as a Stanford type B dissection, was observed (figure 1-2).

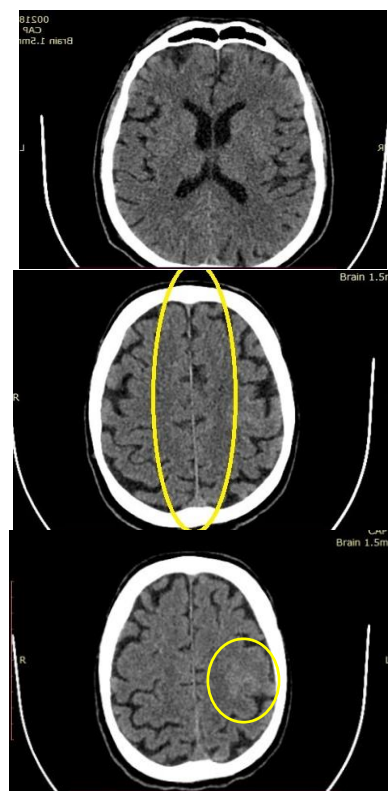


Figure 1. Computed tomographic appearance of brain lesions: a discreet subcortical left frontal hyperdense area measuring approximately 17/18 mm with mild perilesional vasogenic oedema at the cerebral level

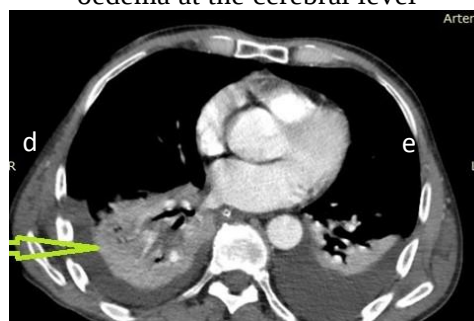




Figure 2. Computed tomographic appearance: a – Right basal posterior pulmonary iodophilic consolidation with air bronchogram at the thoracic level and posttraumatic bilateral pleurisy at the

pleural level; b - In the left hepatic lobe segment II, two hypodense areas measuring 12 mm and subcapsularly measuring 6 mm were identified, possibly indicating a path of dilaceration; c, d – pancreatic suture slice at the level of the pancreas fracture; e, f, g - At the level of the abdominal aorta, a dissection of the abdominal aorta from the origin of the superior mesenteric artery with an extension of approximately 30 mm to the emergence of the left renal artery, classified as a Stanford type B dissection and retroperitoneal hematoma.

Reassessed almost daily, amylasemia ranged between 428.03 and 71.69 U/L (N: 15-125), leukocytes between 15660 and 5160/mm³, and hemoglobin between 12 and 8 g/dL (figure 3-6).

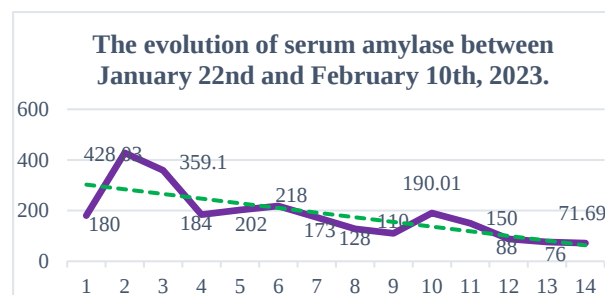


Figure 3. The evolution of serum amylase between January 22nd and February 10th, 2023.

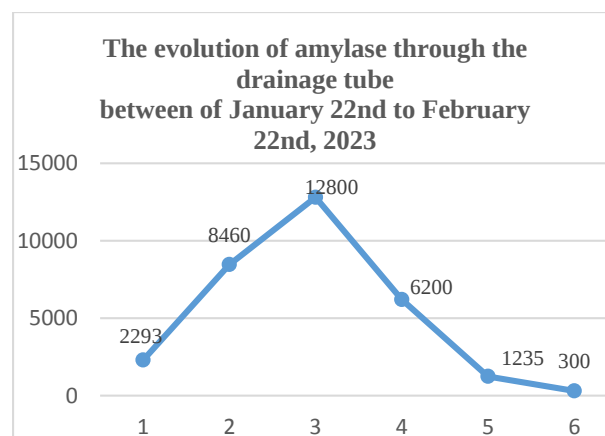


Figure 4. The evolution of amylase through the drainage tube between of January 22nd to February 22nd, 2023

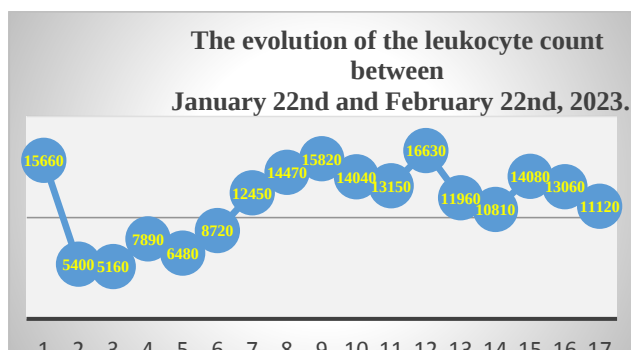


Figure 5. The evolution of the leukocyte count between January 22nd and February 22nd, 2023.

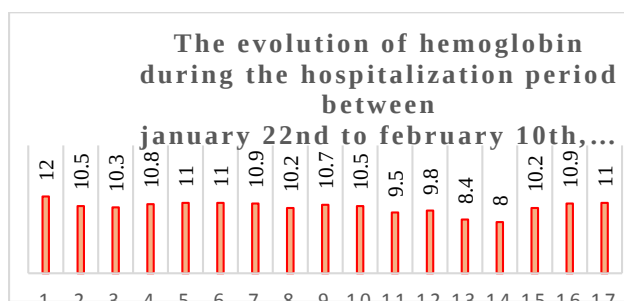


Figure 6. The evolution of hemoglobin during the hospitalization period between January 22nd to February 10th, 2023.

Samples were collected from the fluid externalized through the drainage tubes of the pancreatic lodge at 72 hours, and an amylase level of 2293 U/L was detected. Subsequently, it increased, reaching 12800 U/L, then continuously decreased, reaching around 300 U/L after approximately 4 weeks.

At the same time, daily serum analyses were collected, which showed that the level of serum amylase was consistently decreasing upon discharge, reaching 71.69 U/L, indicating a favourable progression. The drainage from the tubes placed in the large abdominal cavity was 500 mL at 24 hours with a serosanguinous aspect, later decreasing to 250 mL/day at 72 hours. Additionally, diuresis increased from anuria at the time of intervention to a diuresis of 2,200 mL/day at 72 hours post-intervention, indicating a recovery of renal function and proper balance. Blood glucose level was a parameter that was

difficult to balance, showing significant fluctuations (see the graph below – figure 7).

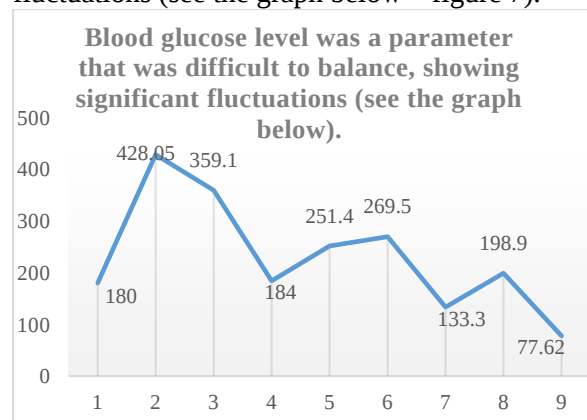


Figure 7. Blood glucose level was a parameter that was difficult to balance, showing significant fluctuations (see the graph below).

The diagnosis at 72 hours after the initial injury includes: **Traumatic dilaceration rupture of the body of the pancreas. Hemoperitoneum. Haemorrhagic shock. Intestinal perforation. Massive retroperitoneal hematoma. Left hepatic lobe hematoma with a small hepatic rupture (of the Glisson's capsule). Severe frontal right-sided trauma. Traumatic abdominal descending aortic dissection. Moderate post-haemorrhagic anaemia. Acute functional renal failure. Cirrhosis of the liver. Post-seizure status. Epilepsy.**

The dynamic evolution of biological samples revealed only a slight increase in serum amylase (428.03 U/L), which occurred at 24 hours from the time of intervention, with its augmentation in the drainage fluid from the pancreatic lodge (up to a maximum of 12800 U/L with subsequent steady decrease) and the initial rise in urinary amylase (up to a maximum of 11522 U/L with subsequent decrease – figure 8).

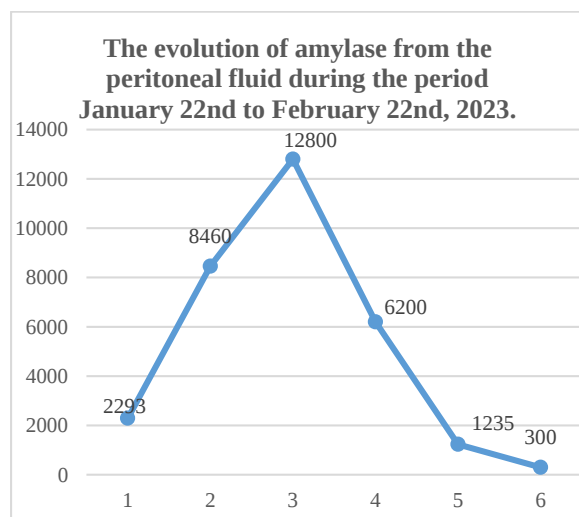


Figure 8. The evolution of amylase from the peritoneal fluid during the period January 22nd to February 22nd, 2023.

TGP (a liver enzyme) presented insignificant variations, while the leukocyte count rose to 16630/mm³, indicating the moment of intestinal volvulus with necrosis, at which point surgical intervention was performed, involving lateral-to-lateral enterectomy with anastomosis. Pancreatic enzymes reached 176 U/L on the fifth day, after which they began to slowly and progressively decrease. Contrary to the evolution of the biochemical profile, the patient's general condition showed an upward trend. On the third day, the patient was advised to sit up and engage in minimal mobilization near the bed. On the fifth day, passage of gas was resumed, and the patient was allowed a liquid oral diet, followed by the reintroduction of solid food on the seventh day. When oral feeding was reintroduced, a slight increase in drainage through the drainage tubes was observed, reaching 400 ml on all tubes. The transfer from the intensive care unit to the surgical ward took place on the 11th postoperative day, with stable cardiovascular and hemodynamic status (aortic dissection being monitored and treated with cardiac medication to maintain blood pressure at 100-110 mmHg and pulse rate at 70-80 bpm), normal respiratory function, afebrile status, normal intestinal transit, and tolerance to liquid

oral feeding. However, on the fourteenth day, intestinal content began to be exteriorized through the drainage tube in the right flank. As a result, an abdominal computed tomography was performed, revealing a volvulus of the intestine around a drainage tube, limited necrosis, loop perforation, localized stercoral peritonitis. Subsequently, a limited enterectomy with lateral-to-lateral enteroenteral anastomosis was performed, along with the repositioning of the drainage tubes. The progression was favourable, with rapid reduction in drainage volume after the re-intervention, and the volume of drainage reaching a level of around 200 mL/day. The volume of pancreatic secretions decreased daily to 50-100 mL on the 21st day, parallel to the overall improvement in the patient's condition following an appropriate diet. Normal intestinal transit was observed, and the patient was allowed oral intake of liquid foods, including milk, fish, poultry, and eggs.

The patient received postoperative medical treatment, including transfusions, antibiotics, analgesics, anticoagulants, and electrolyte rehydration. The evolution was favourable until the fourteenth day post-surgery. On the fourteenth day, when the patient began to externalize intestinal content through the drain tube and around it, indicating stercoral peritonitis caused by ileal volvulus around a necrotic and perforated drain tube.

A subsequent surgical intervention was performed under general anaesthesia through a median laparotomy. During this intervention, multiple postoperative peritoneal adhesions were identified, along with localized stercoral peritonitis. It required the resection of an intestinal loop and a side-to-side anastomosis in two planes. The drain tubes were repositioned. The patient's postoperative recovery after the second surgery was favourable. They were discharged after 31 days with relatively good general condition, stable hemodynamic, respiration, and neurological status.

At the time of discharge, the patient had an external pancreatic fistula with a discharge of approximately 80-100 mL and the appearance of the pancreas is presented in figure 9. Over time, the fistula gradually decreased and eventually

resolved after one and a half months, leading to the removal of the drain tube.



Figure 9. Computed tomographic aspect of pancreas at discharge after 30 days.

At two and a half months post-surgery, the patient's general condition was good, with normal digestive tolerance, no drain tubes, no fistula, and normal mobilization. They were being monitored neurologically and psychiatrically.

The discharge diagnoses were: **Traumatic dilaceration rupture of the body of the pancreas. Hemoperitoneum. Haemorrhagic shock. Intestinal perforation. Massive retroperitoneal hematoma. Left hepatic lobe hematoma with a small hepatic rupture (of the Glisson's capsule). Severe frontal right-sided trauma. Traumatic abdominal descending aortic dissection. Moderate post-hemorrhagic anaemia. Ileal volvulus with limited necrosis. Localized stercoral peritonitis. Acute functional renal failure. Cirrhosis of the liver. Post-seizure status. Epilepsy.**

Discussions

When faced with an abdominal trauma, we need to ask a series of questions that require quick answers. The first question would be: What was the energy of the trauma to which the patient was exposed? Depending on its intensity, it can cause injuries to deeply located organs, as in the present case. The second question: Which organs are affected and what associated injuries have occurred? It is very difficult to perform investigations in the case of a polytraumatized patient in a general state like

the present one. The third question: What explorations can help us, and do we have time to perform them? The fourth question: What therapeutic measures are suitable for the discussed case?

From an epidemiological point of view, we are facing a rare trauma that has caused an exceptional injury, with the incidence being highly variable in statistics; generally, it ranges from 4-5% of abdominal traumas, but there are authors who report 10%. However, on large statistics, pancreatic traumas represent 0.2-1% of total traumas in general. The frequency with which the pancreas is affected is low, being the 10th organ affected in traumas, but the resulting injuries are very severe, leading to high morbidity and mortality (3).

Abdominal contusions involving the pancreas account for about 37% of total injuries, with the remaining being penetrating abdominal injuries. Mortality in traumatic pancreatic injuries exceeds 40% and can reach 50%, while morbidity exceeds 90%. Additionally, in cases of abdominal trauma with pancreatic injury, associated injuries are found in approximately 93% of cases (5, 6, 7, 8, 9, 10, 11).

Regarding aetiology, as mentioned above, the types of injuries depending on the mode of occurrence are either abdominal contusions or penetrating abdominal injuries. Abdominal contusions, in order to cause pancreatic injuries, require a high-intensity energy factor that crushes it against the spinal column, which explains the very high rate of associated injuries. Road accidents are the most commonly involved in these injuries when referring to adults, while for children, the most frequent causes are bicycle accidents.

The clinical or ultrasound diagnosis of traumatic pancreatic ruptures is difficult, regardless of whether it is a contusion (table I) or a penetrating injury (5, 11).

Table I. Computer tomographic signs of pancreatic contusion injury (6, 11).

Specific sign
Complete rupture of the pancreas
Pancreatic laceration
Focal or diffuse pancreatic enlargement/edema
Pancreatic hematoma
Active bleeding/contrast extravasation
Separation of the splenic vein by the posterior face of the pancreas by liquid content
Nespecific sign
Inflammatory changes in peripancreatic and mesenteric fat
Fluid surrounding the superior mesenteric artery
Thickening of the left anterior renal fascia
Acute pseudocyst formation/peripancreatic fluid collection
Fluid in the anterior and posterior pararenal spaces
Fluid in transverse mesocolon and lesser sac
Haemorrhage into peripancreatic fat, mesocolon and mesentery
Extraperitoneal fluid
Intraperitoneal fluid

Penetrating abdominal injuries are caused by firearms or sharp weapons, and associated injuries are encountered in all cases, usually affecting multiple organs (table II). Considering the anatomy of the pancreas, which is located transversely in the abdomen, the impact zone is large, and the injuries can be located in the head, body, or tail of the pancreas, but they can also involve the duodenum, bile ducts (especially in cephalic locations), and pancreatic ducts. Pancreatic duct injuries should be treated as early as possible. The treatment of traumatic pancreatic injuries is associated with a very high rate of complications (exceeding 90%) due to the associated injuries to the pancreas and other organs. Complications can be early or late; early complications typically involve hemorrhage, while the most frequent late complication is pancreatic fistula, but other complications such as peritoneal suppuration and pulmonary thromboembolism are also encountered with high frequency (6, 12, 13, 7, 10, 11, 5, 14, 15).

Table II. Computed tomographic grades of pancreatic gunshot injury (6, 11, 12, 13).

Computed tomographic grades encountered in pancreatic gunshot injury
Grade A Pancreatitis and/or superficial pancreatic laceration at any level
Grade B
B _I Deep laceration in the tail of the pancreas
B _{II} Transection at the level of the tail of the pancreas
Grade C
C _I Deep laceration at the level of the proximal pancreas
C _{II} Transection at the level of the proximal pancreas

Pancreatic rupture is classified as a severe abdominal injury with two major immediate consequences: significant haemorrhage and severe enzymatic peritonitis (evidenced by signs of peritoneal irritation observed during the emergency department examination, such as generalized muscle guarding, limited abdominal respiratory excursion, and paralytic ileus with absence of bowel sounds (2).

Pancreatic injuries are classified into several grades based on their severity, and the most commonly used classification, which is highly important for determining the therapeutic approach (conservative or surgical intervention – table III), is presented below (6, 12, 8, 10, 16, 17).

Henri Mondor, a renowned French surgeon, emphasized the importance of not overlooking a severe injury in cases of abdominal trauma, stating that "death can occur within a few minutes due to internal haemorrhage from the rupture of major vessels, or within two to three days due to generalized peritonitis" (2). In the case of the patient presented, they were initially exposed to high-energy trauma, followed by repeated trauma during seizures with lower energy but resulting in serious abdominal injuries, particularly affecting the pancreas. The crushing force acted perpendicularly on the abdominal wall, causing the pancreas to be compressed against the vertebral column, similar to being trapped in a clamp repeatedly. The exact type of trauma is not precisely known, but according to the

family's account, the patient was found approximately 5-6 hours later. The patient, who had epilepsy, experienced a seizure while being alone at home. During the clinical examination, traumatic injuries to the frontal region and

upper abdominal area were identified, suggesting that the mechanism of injury was repeated abdominal trauma during convulsive episodes.

Table III. Classification of pancreatic trauma and therapeutic options (5, 11, 12, 13, 17).

Grade	Lesions description	Therapeutic options
Grade I	Minor pancreatic contusion without pancreatic duct injury	Preoperative or intraoperative ERCP Preoperative MRCP
Grade II	Large pancreatic contusions without ductal injuries	Conservative procedures are recommended
Grade III	Distal pancreatic ruptures, partial or complete, accompanied by pancreatic duct injuries	Conditional surgical intervention depending on the damage to the duodenum. Due to limited information there are no clear recommendations for pancreatectomy. The use of octreotide is not recommended because it does not bring any benefit. As for routine splenectomy, there is no recommendation.
Grade IV	Proximal pancreatic tears associated with pancreatic duct and pancreatic ampulla injuries	
Grade V	Massive ruptures of the pancreatic head	

Obtaining information through medical history was challenging as the patient was postictal and confused. The presence of haemorrhagic shock and peritoneal signs further complicated the examination, and the family provided ambiguous and inconclusive information. Given the haemorrhagic shock, pre-renal acute renal failure, and severe cardiovascular instability, the patient was promptly transported to the operating room, where specific surgical measures were taken while ensuring fluid and electrolyte balance and acid-base equilibrium.

Shock in this case was determined by multiple mechanisms, including:

a. Vascular injuries leading to a large retroperitoneal hematoma that stimulated the celiac plexus, followed by the activation of a cascade of vasomotor disturbances. The plasma kinins activated by pancreatic enzymes also contributed to this effect, although to a lesser extent in this case due to the relatively quick intervention, resulting in low levels of amylase in the blood. The effect of plasma kinins typically subsides within 24 hours (2).

b. Direct blood loss from the vascular bed, amounting to 2700 mL at the time of the intervention, resulted in a significant decrease in blood pressure. This triggered significant catecholamine release, which, in turn, stimulated an increase in heart rate, leading to cardiovascular instability.

When faced with such a patient, one typically considers all possible injuries, although severe pancreatic injury is not commonly the first consideration. During the FAST (Focused Assessment with Sonography for Trauma) ultrasound examination, the pancreatic injury was only discussed as a possible injury, but the conditions under which the ultrasound was performed were very challenging. The massive retroperitoneal hematoma, hemoperitoneum, and marked secondary pneumoperitoneum made it difficult to identify an obvious pancreatic injury. The generalized guarding, large quantity of fluid in the abdominal cavity, and the presence of blood led me to consider injuries to the spleen (despite intact splenic capsule on ultrasound), liver, and mesentery rupture.

Once exploratory laparotomy was performed, the primary identified lesion was a complete traumatic rupture of the pancreas along the contour of the vertebral column to the left of the superior mesenteric artery (SMA). The incision pathway was vertical, and additional associated injuries, as described earlier, were identified intraoperatively and subsequently confirmed by postoperative CT scan, ultimately leading to the final diagnosis. Key elements to consider during intra-abdominal exploration that guide toward pancreatic injury include retroperitoneal hematoma extending to the root of the transverse mesocolon and/or mesentery, blood in the omental bursa (present throughout in this case), hematoma in the greater omentum, and periduodenal hematoma. Correct surgical management should adhere to Ballinger's Rule of 3 R's: recognition of the diagnosis, resuscitation, and surgical repair through urgent laparotomy.

If an emergency CT scan had been available, several questions would have arisen:

1. Would we have had enough time to perform the exploration considering the patient's overall condition at the time of presentation?
2. Would the associated intra-abdominal injuries have been observed during the urgent examination?
3. Would the pancreatic injury have been detected (considering that statistics indicate that up to 55-60%, or even 70%, of pancreatic injuries can appear normal if explored within the first 24 hours)?

Possible local complications in the postoperative period for patients with such injuries include acute necrotizing hemorrhagic pancreatitis, local suppuration with abscess formation, pancreatic fistula, and posttraumatic pseudocyst.

In cases of severe blunt abdominal trauma with major pancreatic injury, without surgical intervention within 24 hours, the morbidity and mortality rates significantly increase (6). Surgical interventions can range from simple

drainage, pyloric exclusion, duodenal diversion, to more severe cases requiring a Whipple procedure (10). If the patient is hemodynamically unstable, procedures aimed at hemorrhage control are performed initially, followed by re-intervention during the complication period to carry out any necessary resections (6).

The Pancreatic Injury Mortality Score (PIMS) was proposed by the Eastern Association for the Surgery of Trauma in 2016. It provides therapeutic recommendations based on the severity of pancreatic injuries and is validated and ideal for predicting mortality in traumatic pancreatic injuries. The variables used in calculating the score are presented in table IV, V5 (6).

Table IV. Mortality risk score in pancreatic trauma (6).

	Risk	Risk score	Mortality (%)
1	Low	0-5	1
2	Medium	5-9	15
3	High	9-20	50

Table V

. The calculation of the risk score is done according to the grid below (6).

1	Age (over 55)	Yes – 5 points	No – 0 points
2	Shock on admission	Yes – 5 points	No – 0 points
3	Major vascular injuries	Yes – 2 points	No – 0 points
4	Associated injuries	Yes 1 lesion – 1 points 2 lesions – 2 points 3 lesions – 3 points 4 lesions – 4 points Each lesion is equivalent to one point	No – 0 points

Conclusions

In emergency surgical practice, we encounter extremely diverse situations, but traumatic pancreas injury is actually very rare, being an unusual and difficult-to-diagnose traumatic lesion. Computed tomography is the diagnostic method of choice, and MRI with MRCP is used to visualize the pancreatic duct when there is a strong suspicion of Wirsung duct or common bile duct injury. Many surgeons may not encounter a traumatic pancreatic injury throughout their career (in fact, they probably don't want to). The dramatic nature of the case did not allow for patient exploration, as the patient was in an extremely critical condition, necessitating immediate transport to the operating room for specific surgical measures. The surprise of such a lesion was significant, although the mechanism by which it occurred did not suggest it. Statistically speaking, when dealing with trauma, any organ injury can be expected when opening the abdomen of a hemodynamically unstable patient (very low blood pressure of 60/35 mmHg, pulse rate of 140/min, in the presence of trauma that occurred approximately 5-6 hours ago does not leave much room for exploration). In this case, there was zero time available for exploration, except for a FAST ultrasound examination in the emergency department. Generally, when intervening in a traumatic abdomen, the range of possible organic abdominal injuries that we expect includes the most exposed organs (spleen, liver, intestines, kidneys, mesentery, urinary bladder). As presented, the pancreas ranks tenth in terms of frequency of injury. Even though traumatic pancreatic injuries occur in a small percentage, we must not forget about them and consider them as part of the possible spectrum of injuries. Emergency imaging often provides limited diagnostic information, and clinical elements are what practically determine the indication for laparotomy and guide the surgical approach towards the idea of organ rupture.

Additional Information

Patient consent was obtained for presenting this case in a medical publication to contribute to the expansion of the database on this pathology.

The ethics committee of the medical unit

has approved the presentation of the case in a medical article in a medical publication while respecting personal data.

Regarding conflicts of interest, all authors have declared that they have not received any financial support from any organizations related to the published work.

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Other information: All authors have declared that there are no other relationships or activities that have influenced the appearance of the published work.

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